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AIR AND NITROX SATURATION DECOMPRESSION: A REPORT OF 4 1/1
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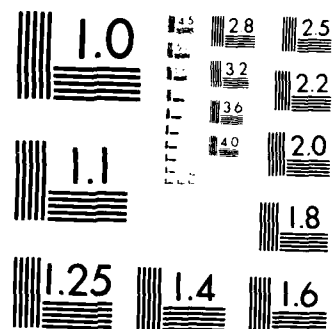
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AIR AND NITROX SATURATION DECOMPRESSION:

A report of 4 schedules and 77 subjects

by

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RUNNING HEAD: Nitrox saturation decompression

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ABSTRACT

Seventy-seven subjects were decompressed from air or nitrogen-oxygen (nitrox) saturation exposures at 18.3 to 40.2 meters sea water (msw) (60 to 132 feet sea water (fswg)) using four different decompression schedules. A 20 hour schedule for decompression from an air saturation-excursion profile at 18.3 msw (60 fsw) resulted in pain-only decompression sickness (DCS) symptoms in 2 of 23 subjects. A 32 and 35 hour schedule from a different air saturation profile at 19.8 and 22.9 msw (65 and 75 fsw) respectively, resulted in DCS symptoms in 1 of 24 subjects. A third and fourth schedule for air or nitrox saturation at 40.2 msw (132 fsw) resulted in DCS symptoms in 3 of 12 and 1 of 18 respectively. No serious (type II) symptoms were observed as a result of any of the decompressions. All DCS cases consisted of knee pain occurring either in the last 3 msw of the decompression, or shortly after surfacing. Doppler ultrasound monitoring revealed venous gas emboli (VGE) in several subjects, but generally only shallow to 6.1 msw (20 fsw). Results demonstrate an overall DCS incidence of 9%, and all cases were pain-only and localized to the knee. The third schedule (US Navy heliox saturation decompression schedule) appears to produce a higher incidence of DCS than the other schedules when used in air or nitrox exposures. Differentiation between the schedules designed for nitrox was impossible due to the limited number of subjects in each and the variable nature of the exposures.

INDEX TERMS: diving; humans; decompression; inert gas; saturation.

INTRODUCTION

A confusing array of decompression schedules for saturation on air or nitrogen-oxygen (nitrox) mixtures at increased ambient pressure has been formulated and tested over the past 20 years (1-9). The incidence of decompression sickness (DCS) has varied widely depending on individual susceptibility and characteristics of the particular exposure. Attempts to apply existing data to decompression schedule formulation have been complicated by widely varying pressurization, excursion and atmosphere profiles. Moreover, since much decompression information never reaches publication, subsequent analysis must rely on hearsay or inadequate data. To formulate sound schedules, and more thoroughly understand the physiology of saturation decompression, a large number of decompression trials with a variety of environmental conditions is required. To this end, this report is a detailed description of the conditions and results of air saturation decompressions occurring at this laboratory in the course of experiments over the past 7 years. This is not intended to be a comprehensive investigation into the theory and physiology of air saturation decompression, nor is it intended to promote the use of the described schedules. Rather, it is intended to add to the body of knowledge of how humans tolerate decompression from air saturation under a variety of conditions.

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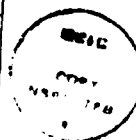
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MATERIALS AND METHODS

Subjects

The subjects for these exposures were active duty or reserve Navy divers. Subjects were not used more than once and no subject had been exposed to elevated pressure for at least 2 weeks preceeding the saturation exposure. Subject physical characteristics for each series of experiments are shown in Table 1. No significant differences in these characteristics existed between the groups of subjects.

Facility

All saturation exposures were performed in the main hyperbaric chamber of the Environmental Simulation Facility located at the Naval Submarine Medical Research Laboratory in Groton, Ct. The chamber was of double lock design, steel construction and was man certified to 107 msw (350 fsw). The diameter measured 2.8 m, the inner lock being 4.6 m in length and the outer 3 m. Separate life support systems for each lock controlled CO₂, temperaure and humidity. CO₂ was monitored continuously in each lock by Beckman 864 analyzers and averaged $0.06 \pm .02\%$ for all exposures. Oxygen was monitored by Beckman 755 analyzers and was maintained at plus or minus 1% of the desired value by Teledyne 323 controllers. Temperature was adjusted to subject comfort and averaged 25.3 ± 0.9 degrees Celsius. All travel during decompression occurred in 0.3 msw (1 fsw) increments. Diet was not

controlled or limited. Sleeping habits were generally not altered. Medications were discouraged and rarely used. Occasionally, acetaminophen, topical antifungal preparations, antacids, pseudoephedrine and topical decongestants were used. Analgesics were not administered during decompression. Activity levels generally consisted of unhurried movement about the chamber (exercise studies discussed below). Scientific procedures consisted of spirometry, other non-strenuous breathing tests, blood draws, special sensory tests (vision, hearing), psychomotor tests, EEGs, ECGs and the like. No oxygen breathing was included in any of the decompression schedules.

Decompression monitoring

Adequacy of decompression was gauged primarily by reported and elicited symptomatology. Symptoms were divided into conventional categories: pain only symptoms (type I) and multisystem or serious symptoms (type II). Treatments were individualized, but generally followed the guidelines of the US Navy Diving Manual (10). Only symptomatic subjects were treated. Treatment details are described with the results. Precordial monitoring for right heart venous gas emboli (VGE) using doppler ultrasound was carried out at regular intervals (every 3 to 6 msw) during the decompression in schedules 2 and 4 only. A Sodelec D.U.G. unit with probe was used for the signal generation, and the Kisman-Masurel scoring scheme (11) was used for analysis of the signals. In this system, two scores are reported - one representing the VGE score with the subject standing at rest, and the other after a series of three deep knee bends (rest/movement). Mean

scores were calculated for each depth, including only those subjects in whom VGE were detected.

Statistics

Schedules were compared on the basis of the presence or absence of DCS symptoms. Two by 2 contingency tables for all combinations were analyzed by either the Fisher test (12) (for small sample size) or the chi-square test (for large sample size).

SCHEDULE DESCRIPTION AND RESULTS

Schedule #1

The decompression schedule is shown in Table 2¹. This schedule is based on the NOAA 3202 M-value matrix, assuming that the 480 minute half time tissue is the rate limiting compartment (2). This decompression was used for two series of air saturation exposures (AIRSAT 1 and 2), with a total of 23 subjects. The storage depth was 18.3 msw (60 fsw), and daily excursions to 30.5 msw (100 fsw) or 45.7 msw (150 fsw) were made as shown in Fig.1A & B. Fifteen hours and 45 minutes elapsed between the 30.5 msw excursions and 19 hrs and 20 min between the 45.7 msw excursions. On each excursion, the subjects exercised on a bicycle ergometer for 30 minutes at approximately 75% of maximal capacity. No decompression symptoms resulted from any of the excursions in these experiments.

The final decompression began about 44 hours after the final excursion in AIRSAT 1 and 47 hours after the final excursion in AIRSAT 2. Twenty-three subjects decompressed using this schedule. One subject (AIRSAT 1) was classified as having type I decompression sickness. Briefly, this subject noted mild, deep seated right knee discomfort on awakening at 1.8 msw (6 fsw), which then increased to moderate levels on reaching the surface. A similar pain had begun to appear in the left knee by the time recompression therapy was initiated (about 30 minutes after surfacing). Complete relief of the left knee discomfort and 90% relief of the right knee pain was achieved in the first 10 minutes of treatment. To minimize further oxygen stress, the second oxygen period at the 18.3 msw (60 fsw) level was eliminated, and the remainder of a standard U.S. Navy Treatment Table 5 (TT5) was completed. Complete relief of all symptoms with no recurrence was the final outcome.

One other subject (AIRSAT 2) reported a feeling of discomfort in the left knee about 3 hours after reaching the surface, but it resolved after several hours with no treatment. None of the other 21 subjects reported symptoms during or subsequent to the decompression. Doppler monitoring was not performed during this series.

Schedule #2

Schedule #2 was developed using the empirical relationship

$$R = k(PiO_2) \quad (1)$$

where R is the rate of ascent in msw/hr, PiO_2 is the inspired oxygen partial pressure in ATA, and k is 1.83 (6 for R in fsw/hr) (13). Since the breathing media for this schedule was air, the PiO_2 decreased as the depth decreased. To satisfy equation (1), the ascent rate also was reduced. For convenience, the ascent rate was reduced at 3 msw (10 fsw) intervals to the rate required by the lowest PiO_2 in the interval.

Schedule #2 was used for a series of 8 exposures (the SUREX experiments), each with 3 subjects. The atmosphere was air throughout, and excursions to the surface were included (see Fig. 1C & D). Further details of these exposures and the ascending excursions is contained in the references (14). The subjects spent a total of 44 hours at 19.8 msw (65 fsw) (SUREX 1-6, n=18), or 22.9 msw (75 fsw) (SUREX 7-8, n=6), and the decompression began about 20 hours after completion of the final ascending excursion. The excursions represented a significant decompression stress, and most subjects complained of pruritus and had detectable VGE during the surface interval. Four subjects had DCS symptoms (3 type I and 1 type II) during or immediately following the excursions. All subjects were asymptomatic prior to initiating the final decompression, and all were decompressed on the same schedule.

One of the 24 subjects (not one of the 4 with DCS symptoms during the excursions) noticed mild, deep seated left knee pain at about 0.6

msw (2 fsw), which was essentially unchanged on arrival at the surface. Physical exam was entirely normal. A standard TT6 was initiated, and full relief was obtained after the first oxygen breathing period at 18.3 msw. There was no recurrence.

The number of subjects with detectable VGE and the mean VGE score for these subjects is shown in Table 3. In schedule #2, no VGE were detected deeper than 3 msw (10 fsw). The highest VGE score occurred in the subject with diagnosed decompression sickness (rest grade 2/movement grade 4). The other scores were generally very low.

Schedule #3

This decompression schedule used is the U.S. Navy standard helium-oxygen (heliox) saturation decompression schedule (10). Because of the very low incidence of decompression sickness associated with the use of this schedule for shallow heliox exposures, it was believed that it might be sufficiently conservative to allow safe decompression from shallow air or nitrox saturation exposures. The ascent rates are shown in Table 2. Rest stops are an integral part of this decompression. The protocol calls for rest stops (no travel) from 2400-0600 and from 1400-1600 independent of the starting time or depth. Continuous travel occurs at all other times of the day.

This decompression was used exactly as indicated in a series of 4 simulated air and nitrox saturation exposures (AIRCAT 3) each with 3

subjects. The pressurization and atmosphere profile is shown in Fig.1E and Fig.2A respectively. Daily 5 hour no-decompression excursions on air to 60.4 msw (198 fsw) were made from a nitrox ($PiO_2=0.30$ ATA) storage depth of 40.2 msw (132 fsw) on days 2,3 and 4. Eighteen hours and 45 minutes elapsed between the excursions. No decompression symptoms resulted from any of the excursions. Twenty hours after the third and final excursion, an isobaric shift to air occurred. The air exposure at 5 ATA continued for 24 hours, afterwhich the final decompression began (at 1000 on day 6). The chamber reached the surface at 1346 on day 8, for a total decompression time of 51:46; 16 hours of rest stops and 35:46 of travel.

Three of the 12 subjects were classified as having type I DCS. All 3 had left knee discomfort, but the time of onset was somewhat different. Two of the 3 subjects initially noted symptoms at about the 1.5 msw (5 fsw) level, and the other subject noticed symptoms about 7 hours after surfacing. One of the 2 subjects noting symptoms while still under pressure was treated according to guidelines in the US Navy Diving Manual (Vol 2)(10) i.e.; recompressed 3 msw deeper than where symptoms were noted, held for 2 hours while breathing 100% oxygen (cycles of 20 min. on, 10 min. off), and then resumed the decompression schedule. Full relief was obtained in this subject immediately on traveling to 4.3 msw (14 fsw), and there was no recurrence. The other subject with symptoms under pressure was allowed to surface (he did not mention symptoms until this time), and then treated with a TFS, which was modified by eliminating 1 oxygen period at 12.3 msw. This treatment modification was believed prudent since

significant signs and symptoms of pulmonary oxygen toxicity (POT) were present (15). This subject had complete relief 5 minutes into the first oxygen period at 18.3 msw, but he had a recurrence of the knee pain only 30 minutes after surfacing from the treatment. A TT6, modified by shortening the 9.1 msw (30 fsw) oxygen periods to 20 min, then resulted in complete relief with no recurrence, and surprisingly, no further pulmonary symptomatology or decrement in pulmonary function. The third subject with symptoms, which appeared after surfacing, was treated with a TT6 10 hours after surfacing. This TT6 was again modified by shortening all oxygen periods to 15 min and increasing air periods to 15 min, as this subject also suffered significant signs and symptoms of POT (vital capacity drop of ~30%) earlier in the decompression schedule. He had immediate, but partial relief of the knee pain on compression to 18.3 msw. The remainder of the discomfort resolved over the course of the treatment, and there was no recurrence. Doppler monitoring was not performed during these exposures.

Schedule #4

This schedule was also derived using equation (1), with k equal to 1.53 (5 if R in fsw/hr) instead of 1.83. This resulted in slower ascent rates, which were believed to be prudent as the subjects were expected to develop significant signs and symptoms of POT, and there is some evidence which suggests that POT reduces decompression tolerance (16). Slower ascent rates also appear to be necessary for

The exposure for which this schedule was used (AIRSAT 4) is shown in Fig. 1F, and the oxygen profile of the exposure is given in Fig. 2B. Briefly, compression to 40.2 msw on a nitrox atmosphere ($PiO_2=0.30$ ATA) is followed, 12 hours later, by an isobaric shift to air ($PiO_2=1.05$ ATA). No excursions were performed. Forty-eight hours after the isobaric shift, another isobaric shift back to nitrox occurred (now $PiO_2=0.50$ ATA), and the decompression started immediately (at 2200 on day 3). The partial pressure of oxygen was maintained at 0.50 ATA until the chamber oxygen level reached 21% (at 14 msw)), after which, the FiO_2 of 21% was maintained to the surface (decreasing PiO_2). The chamber reached the surface at 1508 on day 6 for a total decompression time of 65:08 with no rest periods.

Eighteen subjects, in 6 separate but identical exposures, have decompressed using this schedule. All subjects had significant signs and symptoms of POT, which is described in detail elsewhere (17). During decompression, one subject had onset of bilateral knee discomfort (left greater than right) on awakening on day 6 at about 3 msw. This discomfort would generally abate before the pressure was again reduced by 0.2 msw (every 58 minutes at this point), at which time it would re-appear. This continued to about 0.6 msw, at which time he was rapidly surfaced and transferred to another chamber. He had rapid resolution of the pain at 18.3 msw on oxygen, and a standard VBN PFB was completed. There was no recurrence. The other subjects in the chamber were allowed to continue the schedule. The symptomatic subject had the highest VBN score of the 18 (rest-1/movement-1) at the

3 msw session, but VGE were undetectable after completion of the TT5. The number of subjects with detectable VGE and the mean VGE scores are shown in Table 3 below. VGE were detected deeper in this schedule, with one subject having low scores at the 15.2 msw (50 fsw) level. It is interesting to note that in those subjects with detectable VGE, the scores did not generally increase as one neared the surface. In fact, some subject's VGE scores decreased from 3 msw to the surface.

DISCUSSION

Meaningful comparison of these schedules is almost impossible due to the relatively small numbers of subjects, the well established variability of DCS, differences in the exposures apart from the decompression schedule, and the binomial nature of the data (18). No significant differences in the DCS incidence between the 4 schedules presented here could be detected.

The overall incidence was about 9.1% (7 of 77), and all of the cases were pain-only, or type I symptoms. Most symptoms began while still under pressure, and the knee was the only site in this series. This is consistent with pressurized caisson experience, where the leg was the most common site in workers exposed to compressed air for prolonged periods of time (19). Similarly, knee pain accounts for the majority of DCS symptoms in heliox saturation decompressions and for exposures of comparable depth, the time of onset is similar (20,21,22). Other symptoms commonly associated with deeper heliox saturation exposures (23), such as vestibular symptoms, were not observed in this series.

Although no statistical differences between the schedules can be shown, for the reasons stated above, schedule #3 appears somewhat suspicious, as it accounted for almost half of the DCS, but only one sixth of the subjects. Similar unsatisfactory results (2 DCS cases of pain-only) were obtained from these ascent rates from air saturation exposures of 10.3 hours at 100 fsw (24). In these data

are combined with the results of AIRSAT 3, and compared to a combination of the other exposures, then schedule #3 carries a significantly higher incidence of DCS than the other schedules (28.6 vs 6.2%, $P < .002$). The explanation for this high DCS incidence probably resides in the purpose of this schedule; the ascent rates were designed for helium, not nitrogen, and helium equilibrates more quickly in most tissues than does nitrogen. Based on these results, it would appear that further application of this heliox schedule in air or nitrox saturation exposures is unjustified.

Another factor which may explain the results of schedule #3 is the timing of the rest periods. In the AIRSAT 3 experiments, the decompression was timed so that the subjects surfaced at 1345, or just prior to the 1400 rest stop. Therefore, the subjects were traveling at the 0.91 m/hr rate since 0600, from about 6.7 msw (22 fsw) all the way to the surface. Should things have been timed so that the 2400-0600 rest stop occurred between 3 msw and the surface, the results of this schedule may have been very different. It seems reasonable that rest stops should be in relationship to depth rather than the time of the day.

Equation (1), from which schedules #2 and #4 were developed, is based upon a retrospective study of many saturation dives. An initial analysis of 579 heliox man-decompressions indicated that the average safe rate of ascent increases linearly with the oxygen partial pressure (13). An extension of this analysis to 1179 heliox man-decompressions (to be published separately) confirmed this

relationship and suggested that the average ascent rate also decreases as the saturation depth increases. Analysis of 160 man-decompressions from air or nitrox saturation dives indicated similar effects, but with slower ascent rates than those for heliox. The value of the constant k in equation (1) was chosen based on these retrospective analyses, and was therefore assumed to depend upon both the depth of the saturation exposure and the inspired inert gas species.

It would appear that the decompression outcome from the schedules calculated with equation (1) is somewhat better than a combination of the results from the other two schedules (2 of 42 vs 5 of 35). The difference, however, is insignificant ($P=0.15$). Even if it were significant, it would be difficult to attribute the difference to the schedule alone, as there are marked differences in the excursion and atmosphere parameters between these two groups of exposures. Furthermore, the value of k chosen for use in equation (1) produced ascent rates which were slower than those of schedules #1 or 3, and it is reasonable that a slightly better decompression outcome would be produced by slower ascent rates. Whether or not this schedule provides any decompression advantage, either in safety or efficiency, awaits further testing. An undeniable advantage of equation (1), however, is its simplicity and flexibility.

Other aspects of these exposures further complicate analysis of the decompression outcome. There is some early evidence in animals which suggests that POT increases susceptibility to decompression sickness (16), and decreases the ability of the pulmonary microvasculature to

filter microemboli (25). Subjects in both AIRSAT 3 and AIRSAT 4 suffered significant signs and symptoms of POT (15,17). Although suitable control data is not available for comparison, it is conceivable that those subjects afflicted most severely with POT may be at increased risk for developing DCS compared to those less afflicted. Therefore, since the degree of POT can be estimated from change in the vital capacity (VC) measurement (26,27), one might expect that those subjects with DCS symptoms would have had greater decrements in the VC than those without DCS symptoms. There was no significant difference, however, in the VC decrements between the DCS and the non-DCS groups reported here. Nevertheless, this crude analysis does not negate the role of POT, since the VC may not be the relevant index, or alternatively, more severe POT may be required to detect an effect.

Not only may POT compromise decompression tolerance, it may also complicate treatment. Since current treatment regimens for decompression sickness call for the use of hyperbaric oxygen, a pre-existing degree of POT may compromise tolerance of the treatment itself. This was believed to account for an unusual case of POT in at least one report (9), and was the basis for modifying the treatment tables used for the cases of decompression sickness described here. It appears, however, that subjects treated with hyperbaric oxygen for decompression sickness suffer no further decrement in pulmonary function. The 6 treated cases of decompression sickness in this report had no significant change in the VC from just before to immediately after the treatment ($4.85 \pm .57$ L versus $4.99 \pm .40$ L). Although this

could reflect success of the treatment schedule modifications, it more likely suggests that the effect of an oxygen treatment table is minimal, once recovery from POT has started.

The presence or absence of excursions, either ascending or descending, must also be considered in the interpretation of saturation decompression outcome. Since 44 hours elapsed from the final excursion to the beginning of the decompression in AIRSATs 1, 2 & 3 (schedules #1 and 3), it seems unlikely that this factor would significantly affect decompression outcome, especially since no symptoms of decompression stress were present in any of the subjects after the excursions. Although acclimatization to decompression stress as a result of the repetitive excursions is a consideration (19), a benefit from brief "bounce" exposures for a subsequent saturation decompression has never been demonstrated. Symptoms of DCS did occur on the ascending excursions in the SUREX exposures (schedule #2), and although only 20 hrs elapsed from the last excursion to the start of decompression, there appeared to be no correlation of the excursion results with the decompression outcome. Nevertheless, since little evidence exists to establish the effect of excursions on saturation decompression outcome, the presence of different excursions in the exposures described here remains a confounding factor in the interpretation of these results.

Sufficient precordial doppler monitoring for VGE was not performed to allow correlation with symptoms. However, the only subjects with type 1 DCS from schedules #2 and #4 also had the highest VGE score for

those schedules. Overall, quantities of VGE were very low. The deeper exposures had a tendency to produce detectable VGE earlier than the shallower exposures, as would be expected.

In conclusion, 7 cases of decompression sickness out of 77 decompressions from nitrox saturation exposures at depths of 18.3 to 40.2 msw are described in detail. The overall DCS incidence rate was about 9%, and was similar in character and timing with that reported for heliox exposures. The US Navy heliox saturation decompression schedule, when used for air or nitrox saturation exposures, appears to produce a higher incidence of DCS than schedules designed for nitrox. Differentiation between the schedules designed for nitrox saturation exposures was impossible due to the relatively small number of subjects, the difference in exposure parameters, and the binomial nature of the data. Many more decompressions using uniform, uncomplicated criteria and procedures, and incorporating more quantifiable indexes of decompression stress, will be necessary before sound concepts of air and nitrox saturation decompression can be formulated.

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REFERENCES

1. Workman RD. Calculation of the air saturation decompression tables. Navy Experimental Diving Unit Research Report 11-57, 1957.
2. Hamilton RW, Kenyon DJ, Frietag M, Schreiner HR. NOAA OPS I and II. Formulation of excursion procedures for shallow undersea habitats. Technical Memo. UCRI No. 731, Union Carbide Corporation, Tarrytown, New York. 1971.
3. Hamilton RW. Development of decompression procedures for depths in excess of 400 feet. The ninth Undersea Medical Society workshop. Undersea Medical Society, Inc. Report No. WS: 2-28-76, 1976.
4. Miller JW, Lambertsen CJ. Project Tektite I: An open sea study of prolonged exposures to a nitrogen oxygen environment at increased ambient pressure. In: Lambertsen CJ. ed. Underwater Physiology. Proceedings of the fourth symposium on underwater physiology. New York: Academic Press, 1971: 551-558.
5. Miller JW, Vanderwalker JG, Waller RA. Scientists in the sea; Tektite II. U.S. Department of the Interior, Washington D.C.
6. Miller JW (ed) Vertical Excursions Breathing Air from Nitrogen-Oxygen or Air Saturation Exposures. U.S. Department of Commerce, Rockville, MD. May 1976.

7. National Oceanic and Atmospheric Administration . NOAA Diving Manual: diving for science and technology. U.S. Government Printing Office, Washington, D.C., 1980.
8. Lambertsen CJ. Decompression from acute and chronic exposure to high nitrogen pressure. Aerospace Med 1973; 44:834-836.
9. Hamilton RW, Adams GM, Harvey CA, Knight DA. SHAD - NISAT: A composite study of shallow saturation diving. Naval Submarine Medical Research Laboratory Research Report No. 985. 1982.
10. U.S. Navy Diving Manual, Vol II, Change 1. U.S. Government Printing Office No. 008-046-00086-7, 1973.
11. Kisman KE, Masurel G, Guillerm R. Bubble evolution code for doppler ultrasonic decompression data. Undersea Biomed Res 1978; 5(Suppl):28.
12. Fisher RA. Statistical methods for research workers. (5th Ed) Edinburgh: Oliver & Boyd, 1934.
13. Vann RD, Dick AP. Prediction of HeO₂ saturation decompression schedules from the results of past dives. Undersea Biomed Res 1981; 3(suppl):4.
14. Ekenhoff RG, Parker JW. Latency in the onset of decompression sickness on direct ascent from air saturation. J Appl Physiol: Respir

Environ Exercise Physiol 1984; 56: (in press).

15. Dougherty JH, Styer DJ, Eckenhoff RG, Hunter WL. The effects of hyperbaric and hyperoxic conditions on pulmonary function during prolonged hyperbaric chamber air saturation dives. Undersea Biomed Res 8(suppl):29, 1981.

16. Flynn ET, Greene KM. Effect of pulmonary oxygen toxicity on decompression tolerance. Undersea Biomed Res 1981; 8(Suppl):49.

17. Eckenhoff RG, Dougherty JH, Messier AA, Gruber BR, Parker JW, Jordan JE. Human pulmonary oxygen toxicity in hyperbaric air. Fed Proc 1984; 43:10034 Abstract.

18. Berghage TE. The probabilistic nature of decompression sickness. Undersea Biomed Res 1974; 1:189-196.

19. Paton WDM, Walder DN. Compressed air illness. Special Report. Medical Research Council No. 281, London.

20. Hanson RG, Vorosmarti J, Barnard EEP. Decompression sickness after saturation diving. In: Shilling CW, Beckett MW, eds, Underwater Physiology VI. Proceedings of the sixth symposium on underwater physiology. Bethesda: Federation of American Societies for Experimental Biology 1973: 537-545.

21. Sarnat JK, Berghage TE. Review and analysis of cases of

decompression sickness occurring under pressure. Navy Experimental Diving Unit Research Report 12-71, 1971.

22. Summitt JK, Kulig JW. Saturation dives, with excursions, for the development of a decompression schedule for use during SEALAB III. Navy Experimental Diving Unit Research Report 9-70, 1970.

23. Greene KM, Lambertsen CJ. Nature and treatment of decompression sickness occurring after deep excursion dives. Undersea Biomed Res 1980; 7:127-139.

24. Thalmann ED, Zumrich JL, Schwartz HJC, Butler FK. Accommodation to decompression sickness in HeO2 divers. Undersea Biomed Res 1984; 11(Suppl): 1-10.

25. Butler BD, Hills BA. Effect of excessive oxygen upon the capability of the lungs to filter gas emboli. In: Bachrach AJ, Matzen MM, eds. Underwater Physiology VII. Proceedings of the seventh symposium on underwater physiology. Bethesda: Undersea Medical Society, Inc., 1981:95-102

26. Clark JM, Lambertsen CJ. Pulmonary oxygen toxicity: A review. Pharmacol Rev 1971; 23:37-133.

27. Wright WB. Use of the University of Pennsylvania Institute for Environmental Medicine procedure for the calculation of cumulative pulmonary oxygen toxicity. Naval Experimental Diving Unit Report No. 2-71, 1971.

FIGURE LEGENDS

1. Pressurization profile for all the exposures described in this report. A: AIRSAT 1 (schedule #1), B: AIRSAT 2 (schedule #2), C: SUREX 1-6 (schedule #2), D: SUREX 7,8 (schedule #2), E: AIRSAT 3 (schedule #3), F: AIRSAT 4 (schedule #4). Clear areas represent air as the breathing media, and shaded areas represent other nitrogen oxygen mixtures; crosshatching - 0.30 ATA oxygen, balance nitrogen; dotted area - 0.50 ATA oxygen, balance nitrogen.

2. Oxygen partial pressure profiles for A) AIRSAT 3 and B) AIRSAT 4. The diluent was nitrogen in all cases. The exposures not shown used air throughout.

FOOTNOTES

1. This schedule is the same one used for the Shallow Habitat Air Dive experiment (9), with the arbitrary 8 hour sleep hold at 3.7 msw deleted.

TABLE 1

SUBJECT PHYSICAL CHARACTERISTICS*

EXPERIMENT	N	AGE (yr)	HEIGHT (cm)	WEIGHT (Kg)	BODY FAT (%)
AIRSAT 1	11	25.5 \pm 5.0	178 \pm 4.0	77.6 \pm 7.0	19.8 \pm 4.5
(schedule #1)		(20-35)	(172-185)	(69.9-91.2)	(14.9-28.0)
AIRSAT 2	12	23.8 \pm 3.3	180 \pm 8.0	77.3 \pm 9.0	16.4 \pm 3.6
(schedule #1)		(21-32)	(162-189)	(61.7-95.7)	(11.0-22.0)
SUREX	24	26 \pm 5.0	175 \pm 6.5	75.8 \pm 8.6	13.3 \pm 4.4
(schedule #2)		(20-38)	(164-191)	(56.5-94.8)	(6.5-24.6)
AIRSAT 3	12	30.1 \pm 3.6	176 \pm 9.0	81.4 \pm 10.0	22.1 \pm 4.3
(schedule #3)		(25-36)	(162-194)	(68.4-104.4)	(15.0-27.0)
AIRSAT 4	18	29.3 \pm 6.9	174 \pm 8.3	78.8 \pm 9.6	14.0 \pm 5.0
(schedule #4)		(19-40)	(160-186)	(61.7-107.4)	(7.6-28.7)

* - units expressed as mean \pm standard deviation, (range).

TABLE #2

SCHEDULES

2/23/52

SURF 65- 1/15
SURF 75- 0/6

3/12

AIRSAT 122

AIRSAT 3

1	2	3	4
DEPTHS*	RATE**	DEPTHS	RATE
18.3-13.7	33 10	22.3-21.3 ^{75 70 t}	49 15
13.7-6.1	49 15	21.3-18.3 ^{70 60 2.53}	56 17
6.1-1.5	108 33	18.3-15.2 ^{60 50}	62 19
1.5-0.0	118 36	15.2-12.2 ^{50 40}	72 22
		12.2-9.1 ^{40 30}	82 25
		9.1-6.1 ^{30 20}	98 30
		6.1-3.0 ^{20 10}	121 37
		3.0-0.0 ^{10 0}	157 43
		40.2-30.5 ^{132 100}	39
		30.5-15.2 ^{100 50}	49
		15.2-0.0 ^{50 0}	66
		40.2-15.2 ^{132 50}	79 24
		15.2-12.2 ^{50 40}	85 26
		12.2-9.1 ^{40 30}	93 30
		9.1-6.1 ^{30 20}	118 36
		6.1-3.0 ^{20 10}	144 44
		3.0-0.0 ^{10 0}	190 53

TOTAL

32:06****

TIME

20:00

34:46

51:42***

65:08

Notes:

* In meters sea water (msw).

** In minutes per msw.

*** Includes 16 hours of rest stops (see text).

**** Total time for schedule from 19.8 msw.

TABLE #3

VENOUS GAS EMBOLI SCORES ON ASCENT

	Depth, msw (fswg)					
	15.2 (50)	9.1 (30)	6.1 (20)	3.0 (10)	1.5 (5)	0
<hr/>						
<u>SCHEDULE #2 (N=24)</u>						
No. subjects with VGE	0	0	0	1	5	5
Mean VGE score*	0/0 0	0/0 0	0/0 0	0/1 .24	.4/1.9 .4	.4/2 .42
<u>SCHEDULE #4 (N=18)</u>						
No. subjects with VGE	1	2	4	4	-	4**
Mean VGE score*	0/1 .06	0.5/1.0 .11	0.8/2.0 .44	0.3/1.3 .29	-	0.5/1.8 .4

* The means include only those subjects with detectable VGE, not all of the subjects.

** The one subject with DCS (see text) was treated before surfacing, and had no detectable VGE after the treatment.

Handwritten notes and calculations:

$\frac{0}{24} = 0\%$
 $\frac{0}{18} = 0\%$
 $\frac{1}{18} = 5.5\%$
 $\frac{2}{18} = 11.1\%$
 $\frac{4}{18} = 22.2\%$
 $\frac{4}{18} = 22.2\%$
 $\frac{4}{18} = 22.2\%$

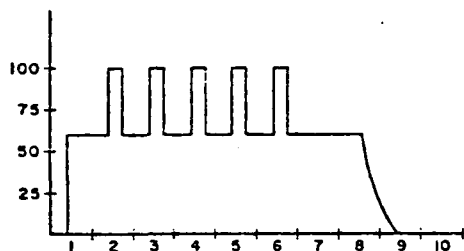
TABLE #4

SCHEDULE CHARACTERISTICS AND RESULTS

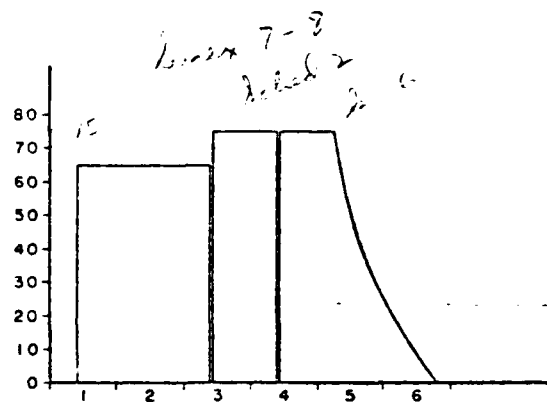
SCHEDULE	SAT DEPTH*	EXCURSIONS*	TOTAL DC TIME	# SUBJECTS	DCS SYMPTOMS
1 (AIRSAT 1&2)	18.3	30.5 & 45.7	20:00	23	2 (8.7%)
2 (SUREX)	19.7 & 22.9	0	32:06 & 34:46	24	1 (4.2%)
3 (AIRSAT 3)	40.2	60.4	51:42	12	3 (25%)
4 (AIRSAT 4)	40.2	NONE	65:03	18	1 (5.6%)

* - Units are meters sea water (msw).

(A)

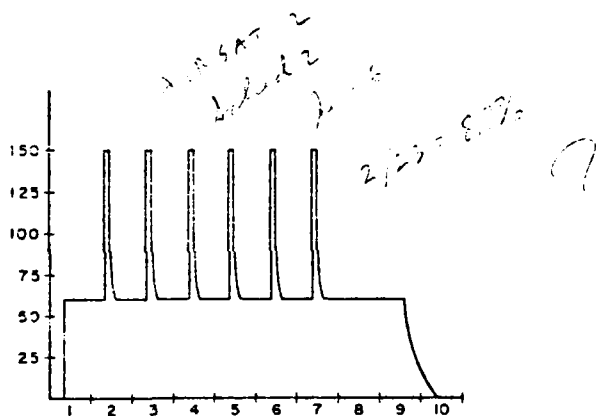


(D)

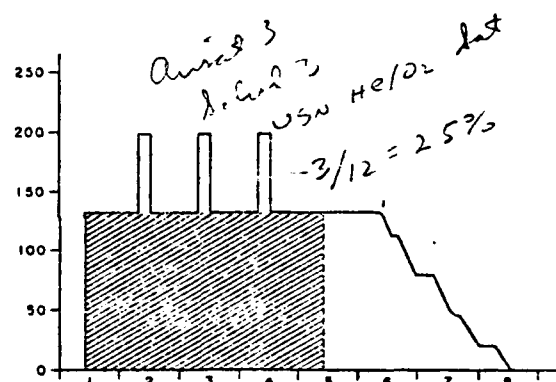


(B)

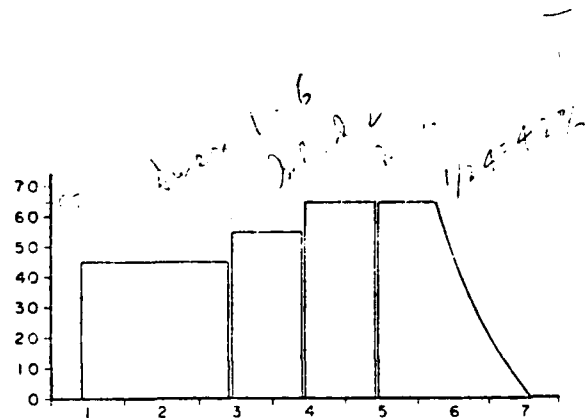
DEPTH FSWG



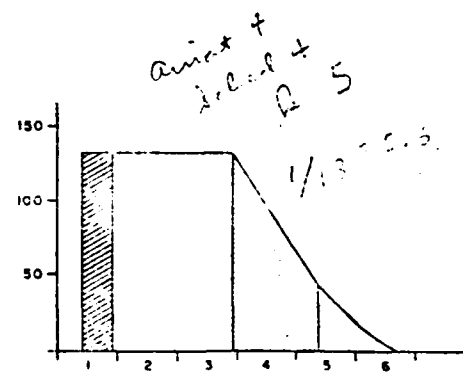
(E)



(C)

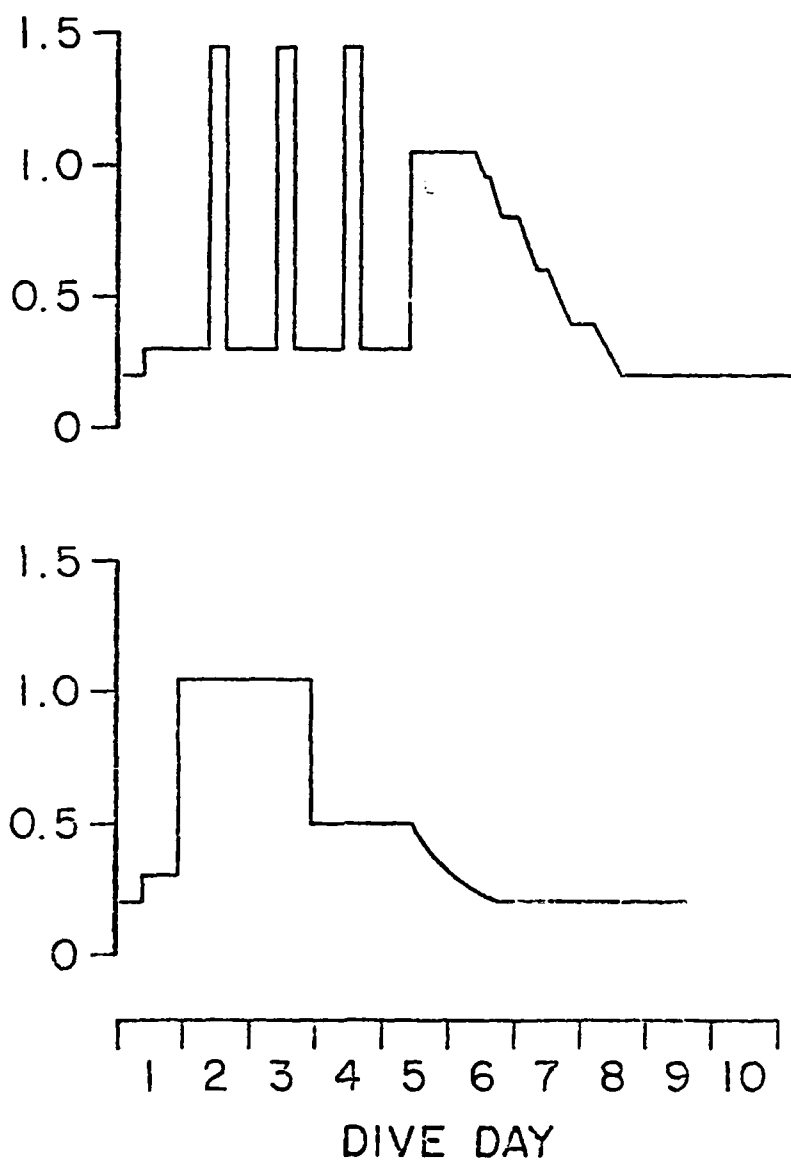


(F)



ELAPSED TIME (DAY)

OXYGEN PARTIAL PRESSURE, ATA



END

FILMED

5-85

DTIC